Appendix D – Toxicity Information for Select Constituents

D.1 Monomethyl Amine Nitrate – Monomethylamine

D.1.1 Derivation of Oral Reference Dose

Monomethylamine nitrate (MMAN) was produced by DuPont for use as a sensitizer with water gel explosive formulations. There are no other industrial applications for MMAN. MMAN readily dissociates in water to monomethylamine (MMA) and nitrate, and is not expected to be persistent in the environment. Current analytical methods do not distinguish MMAN from MMA.

EPA has not published toxicity information or toxicity values (e.g., RfD) for MMAN or MMA. MMAN/MMA are not considered carcinogens by EPA. Chronic studies of the toxicological effects of MMA were not found in the current literature. Evidence from occupational studies have shown no long-lasting health effects when workers were exposed to MMAN via inhalation and dermal contact (ACGIH, 1988).

In the absence of human or animal toxicity dose-response studies, the RfD in the draft final RA was derived using an alternative approach. MMA is a natural ingredient in many foods including vegetables (e.g., average concentration in several different types of vegetables was 21.95 ppm) and seafood (Neurath et al., 1977). The RfD was derived based on the amount of MMA an individual consumes daily via ingestion of vegetables. It is assumed that consuming MMA in the diet does not result in any adverse health effects.

The RfD represents the amount of MMA that the average adult and child consumes daily as part of their normal diet (i.e., vegetables only) and then the dose is determined by dividing these values by the child and adult body weights, respectively. These doses are conservative approximations of the average doses of MMA in the diet because (1) the vegetables considered represent a small part of the American diet which contains other sources of MMA (e.g., seafood), (2) the ingestion rate of vegetables used was a U.S. population average which may underestimate the intake of vegetables and MMA by some groups such as vegetarians, and (3) the measurements of MMA in uncooked vegetables are underestimates of the amount consumed because cooking and canning increase MMA content of foods (Lin et al.,1983).

The derived oral RfD is 0.0175 mg/kg-day for a child and 0.0081 mg/kg-day for an adult. The RfD for the adult is lower (i.e., more protective when used in a risk evaluation) than the RfD for the child because the average adult eats less MMA each day per kilogram of body weight. These RfDs are adequate (protective) for evaluating potential risks associated with human exposure to MMAN or MMA. The lower, more protective RfD (i.e., 0.0081) was selected for the Dupont Works Site and approved by Ecology (PIONEER, 1997).

D.1.2 References

ACGIH (American Conference of Governmental and Industrial Hygienists). 1988. Documentation of the Threshold Limit Values and Biological Exposure Indies. Cincinnati, Ohio. American Conference of Governmental and Industrial Hygienists.

Neurath , G. B. et al. 1977. Primary and secondary amines in the human environment. Food and Cosmetic Toxicology. 15:275-282.

Lin, J.K., Lee, Y.J., and H.W. Chang. 1983. High concentrations of dimethylamine and methylamine in squid and octopus and their implications in tumor etiology. Food and Chemical Toxicology. 21(2):143-149.

PIONEER (PIONEER Technologies Corporation). 1997. Letter from Brad Grimsted to Mike Blum.

D.2 Toxicity Profile for Arsenic

D.2.1 Introduction

Arsenic is a naturally occurring element that is widely distributed in the earth's crust. In the environment, arsenic is combined with oxygen, chlorine, and sulfur to form inorganic arsenic compounds. It is released into the air by volcanoes, the weathering of arsenic-containing minerals and ores, and by commercial or industrial processes (EPA, 2002a). Arsenic is persistent and does not breakdown in the environment. It can only change its form. Once it is released into the air, it will settle to the ground or be washed out of the air by rain. Once in soil, arsenic can be taken up and converted to organic arsenic by plants and animals.

The primary commercial use of inorganic arsenic is as a wood preservative, while organic arsenic compounds are typically used in pesticides (ATSDR, 2001). At the Site, arsenic is most likely present due to its use as a pesticide to control vegetation along the narrow gauge railroad. Speciation of arsenic at the Site has shown it to be present primarily in the inorganic form.

D.2.2 Health Effects

Inorganic arsenic compounds are generally more toxic to humans than organic arsenic compounds. Breathing high levels of inorganic arsenic can cause a sore throat or irritated lungs. Inhalation of lower levels of arsenic over a long time can cause darkening of the skin and appearance of small "corns" or "warts" on the body (ATSDR, 2001). Inhalation of arsenic has also been associated with development of lung cancer (EPA, 2002a).

Ingestion of high levels of inorganic arsenic can cause death, while ingestion of lower levels can cause nausea, vomiting, anemia, abnormal heart rhythm, and circulatory system damage (ATSDR, 2001). Ingestion of inorganic arsenic has been linked to a form of skin cancer and also to bladder, liver, and lung cancer (EPA, 1994). The World Health Organization, the Department of Health and Human Services, and the EPA have determined that inorganic arsenic is a human carcinogen (ATSDR, 2001).

D.2.3 Basis for Toxicity Values Used in the Risk Assessment

Toxicity values for both cancer and non-cancer health effects were used in the RA to calculate remediation levels. The value used to calculate a remediation level based on non-cancer health effects was an oral reference dose (RfD) of 0.0003 mg/kg-day, based on the observance of hyperpigmentation, ketatosis, and possible vascular complications in people exposed to inorganic arsenic in drinking water. The value used to calculate a cleanup levels and remediation levels based on cancer risk was a cancer potency factor (CPF) of 1.5 $(mg/kg\text{-}day)^{-1}$, based on the occurrence of skin cancer in humans exposed to inorganic arsenic in drinking water (EPA, 2002b).

D.2.4 References

ATSDR (Agency for Toxic Substances and Disease Registry). 2001. ToxFAQs for Arsenic, July, 2001.

EPA (United States Environmental Protection Agency). 2002a. Hazard Summary for Arsenic and Compounds. Unified Air Toxics Website. Office of Air Quality Planning & Standards.

EPA (United States Environmental Protection Agency). 2002b. EPA's Integrated Risk Information System Database, 1st Quarter Update, 2002.

D.3 Toxicity Profile for Lead

D.3.1 Introduction

Lead is a naturally occurring metal found in small amounts in the earth's crust. Lead is also present due to human activities such as burning fossil fuels, mining, and manufacturing. Manufacturing uses of lead include the production of batteries, ammunition, metal products, and devices used to shield x-rays (ATSDR, 2001).

Lead does not breakdown in the environment. It can only change its form. When lead is released to the air, it may travel long distances before it settles out and sticks to soil particles.

Because of health concerns, the lead content in gasoline, paints, ceramic products, caulking, and pipe solder has been dramatically reduced or eliminated in recent years (ATSDR, 2001).

Human exposure to lead occurs through a combination of inhalation and oral exposure, with the oral route generally contributing a greater proportion of the dose for the general population. The effects associated with exposure to lead are the same regardless of the route of exposure (inhalation and oral) (EPA, 2002).

D.3.2 Health Effects

Lead effects almost every organ and system in the body. The most sensitive system is the central nervous system, particularly in children, where slow cognitive development and delayed growth have been noted following chronic exposure (EPA, 2002). Lead also damages kidneys and the reproductive system. At high levels, lead may decrease reaction time, cause weakness in fingers, wrists, or ankles, and possibly affect memory. Lead may also cause anemia.

Although there is evidence that lead can cause cancer in laboratory animals, there is inadequate evidence to clearly determine that it causes cancer in humans (ATSDR, 2001).

D.3.3 Basis for Toxicity Evaluation in the Risk Assessment

The EPA has chosen to evaluate potential adverse health effects of lead using a physiologically-based model that takes into account lead consumption through diet and environmental sources such as air, soil, and water. The model used for establishing lead remediation levels in non-residential areas like the DuPont Site is the Adult Lead Model (EPA, 1996). This model estimates fetal blood lead concentrations in women exposed to lead in soil. A developing fetus is considered the most sensitive receptor associated with adult exposure to lead. The soil cleanup levels and remediation levels presented in the RA for lead were based on limiting the fetal blood lead level to 10 ug/dl.

D.3.4 References

ATSDR (Agency for Toxic Substances and Disease Registry). 2001. ToxFAQs for Lead, Updated June 11, 2001.

EPA (United States Environmental Protection Agency). 1996. Recommendations of the Technical Review Workgroup for Lead for an Interim Approach to Assessing Risks Associated With Exposures to Lead in Soil. Technical Review Workgroup for Lead. Adult Risk Assessment Committee.

EPA (United States Environmental Protection Agency). 2002. Hazard Summary for Lead and Compounds. Unified Air Toxics Website. Office of Air Quality Planning & Standards.

D.4 Toxicity Profile for Mercury

D.4.1 Introduction

Mercury is a naturally occurring metal found in the environment. Mercury enters the environment as the result of the normal breakdown of minerals in rocks and soil from exposure to wind and water. Human activities have also resulted in the release of mercury to the environment. Most of the mercury released from human activities comes from the burning of fossil fuels, mining, smelting, and from solid waste incineration (ATSDR, 1999). Mercury can exist in three general forms: as metallic mercury, inorganic mercury, and organic mercury.

Mercury is persistent and does not breakdown in the environment. Once it is released into the air, mercury will settle to the ground or be washed out of the air by rain. Once in soil, mercury combines with other elements, such as chlorine, sulfur, or oxygen, to form inorganic mercury compounds or "salts". Alternatively, mercury deposited on the soil may be taken up by microorganisms and converted to organic mercury (ATSDR, 1999). Metallic mercury is not typically found in the environment.

Exposure to organic mercury is generally only of concern when consumption of fish and other aquatic organisms is considered likely, due to the ability of methyl mercury to concentrate in animal tissues. At the DuPont Site, potential exposure to mercury is through direct contact with soil. Therefore, the focus of this toxicity profile is on the health effects associated with inorganic mercury.

D.4.2 Health Effects

In general, exposure to inorganic mercury is less harmful than exposure to the other forms of mercury because inorganic mercury is less able to reach the brain. Inhalation of inorganic mercury is not associated with adverse health effects. However, ingestion of high levels of inorganic mercury can permanently damage the brain, kidneys, and developing fetuses. Effects on brain functioning may result in irritability, shyness, tremors, changes in vision or hearing, and memory loss (ATSDR, 2001).

Although there is evidence that inorganic mercury can cause cancer in laboratory animals, there is inadequate evidence to clearly determine that it causes cancer in humans (ATSDR, 2001).

D.4.3 Basis for Toxicity Value Used in the Risk Assessment

The toxicity value used to calculate cleanup and remediation levels based on non-cancer health effects was an oral reference dose (RfD) of 0.0003 mg/kg-day. This value was calculated from a study showing immune system effects in rats fed inorganic mercury in their diet (EPA, 2002).

D.4.4 References

ATSDR (Agency for Toxic Substances and Disease Registry). 1999. Toxicological Profile for Mercury. U.S. Dept of Health and Human Services.

ATSDR (Agency for Toxic Substances and Disease Registry). 2001. ToxFAQs for Mercury, Updated June 11, 2001.

EPA (United States Environmental Protection Agency). 2002. EPA's Integrated Risk Information System Database, 1st Quarter Update, 2002.

D.5 Toxicity Profile for 2,4,6-Trinitrotoluene (TNT)

D.5.1 Introduction

2,3,6-Trinitrotoluene (TNT) is a yellow, odorless solid that does not occur naturally in the environment. It is an explosive used in military shells, bombs, grenades, for industrial uses, and in underwater blasting. TNT enters the environment resulting from manufacturing activities, processing and destruction of bombs, and the recycling of explosives (ATSDR, 2001). Once in the environment, it is rapidly broken down by sunlight. It can also be broken down by microorganisms, but this is a much slower process. TNT can accumulate in small amounts in fish and plants, but potential exposure to humans at the DuPont Site is through accidental ingestion of soil.

D.5.2 Health Effects

Workers who were exposed to high airborne levels of TNT during production of explosives experienced health effects such as anemia and abnormal liver function. Other effects seen in humans include skin irritation after prolonged skin contact, and cataract development after more than one year of exposure (ATSDR, 2001).

Although there is evidence that TNT can cause cancer in laboratory animals, there is inadequate evidence to clearly determine that it causes cancer in humans (ATSDR, 2001).

D.5.3 D.5.3 Basis for Toxicity Values Used in the Risk Assessment

Toxicity values for both cancer and non-cancer health effects were used in the RA to calculate remediation levels. The value used to calculate cleanup levels and remediation levels based on non-cancer health effects was an oral reference dose (RfD) of 0.0005 mg/kg-day, based on the observance of liver effects in dogs exposed to TNT in their diet. The value used to calculate a remediation level based on cancer risk was a cancer potency factor (CPF) of 0.03 $(mg/kg\text{-}day)^{-1}$, based on the occurrence of bladder tumors in rats exposed to TNT in their diet (EPA, 2002).

D.5.4 References

ATSDR (Agency for Toxic Substances and Disease Registry). 2001. ToxFAQs for 2,4,6-Trinitrotoluene (TNT), Updated June 11, 2001.

EPA (United States Environmental Protection Agency). 2002. EPA's Integrated Risk Information System Database, 1st Quarter Update, 2002.

D.6 Toxicity Profile for Total Petroleum Hydrocarbons (TPH)–as Bunker C Fuel

D.6.1 Introduction

Total petroleum hydrocarbons (TPH) is a term used to describe a large family of several hundred chemical compounds that originally come from crude oil. Crude oil is used to make petroleum products. These products contain so many individual compounds that it is not practical to quantify each one. Instead, identification is made by performing chemical analysis of a category of TPH, as defined by weight of product. Some compounds that may be found in TPH are hexane, jet fuels, mineral oils, benzene, toluene, xylenes, naphthalene, and fluorine, as well as other petroleum products and gasoline components. However, it is likely that any given sample of TPH will only contain a subset of these compounds (ATSDR, 2001).

TPH may enter the environment through accidental spills, from industrial releases, or as byproducts from commercial or private uses. Once in the environment, certain fractions of TPH may be broken down by microorganisms, while other fractions may move into soil where they may persist for a long time (ATSDR, 2001).

The TPH product used at the DuPont Site was Bunker C fuel. Therefore, the discussion of health effects will pertain to those associated with exposure to this TPH product.

D.6.2 Health Effects

Human contact with Bunker C fuel has been associated with skin irritation. In addition, ingestion can cause nausea, vomiting, diarrhea, and central nervous system effects such as restlessness (U.S. Oil & Refining Co., 1998).

Bunker C Fuel may also contain some polycyclic aromatic hydrocarbons (PAHs), that have been shown to cause skin cancer in laboratory animals, however, there is inadequate evidence to clearly determine that they cause cancer in humans.

D.6.3 Derivation of a Bunker C Cleanup Level

Recognizing that the risk posed by Bunker C is largely attributable to its carcinogenic polycyclic aromatic hydrocarbon (cPAH) components, a Site-specific correlation analyses were performed to assess the statistical relationship between cPAH and TPH concentrations. Thus the Bunker C cleanup level is based on the cPAH toxicity values. See Appendix C for details on how this information was used to derive the cleanup level.

D.6.4 References

ATSDR (Agency for Toxic Substances and Disease Registry). 2001. ToxFAQs for Total Petroleum Hydrocarbons (TPH), Updated June 11, 2001.

U.S. Oil & Refining Company. 1998. Material Safety Data Sheet for Bunker C. Revised August 8, 1998.